Structure of Heart & Blood Vessels

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Human Heart

- **Muscular pump**
- **Pumps → The blood into the blood vessels**
- **Supplies the blood → To the tissues of the body**

Right side of the Heart

- **Supplies blood ➔**
- **To the *Lungs* ➔**
- ***For oxygenation***

Left side of the Heart

- **Supplies oxygenated blood ➔**
- ***To the tissues***

Anatomy & Structure of Heart

- **Human heart weighs ➔ 300 gm**
- **Covered by an**
- **Parietal Pericardium**
- **Visceral Pericardium**

Parietal Pericardium

- **Outer ➔ Fibrous pericardium**
- **Inner ➔ Serous pericardium**
- **Pericardial Cavity ➔ Space between 2 layers**

Pericardial Fluid

- Fluid in ➔ Periocardial cavity
- Ensures smooth movement of heart
- Pericardial fluid ➔Acting as Lubricant

Applied Physiology

- **Pericardial effusion**
- **Some disease conditions**
- **Pericardial fluid increases in Volume**

Cardiac Tamponade

- Due to accumulation of large amount of fluid
- In pericardial cavity
- Massive bleeding due to injury to the heart
- Acute pericarditis

Cardiac Tamponade

- Life threatening condition
- Due to
- Severe reduction in Cardiac output

4 ➔ Chambers of the Heart

- 2 ➔ Atria
- 2 ➔ Ventricles

2 ➔ Atria & 2 ➔Ventricles

- **2 Atria Separated by ➔Interatrial Septum**
- **2 Ventricles separated by ➔ Interventricular septum**

Right Atrium → Receives Blood

- Different tissues in the body
- Deoxygenated Blood
- Through
- Superior Venae cavae (SVC)
- Inferior Venae Cavae (IVC)

Left Atrium → Receives Blood

- From → Lungs
- Oxygenated blood
- Through
- Pulmonary Veins

Right Ventricle ➔ Pumps Blood

- **Pulmonary circulation**
- **Through ➔**
- **Pulmonary Artery (Deoxygenated Blood)**

Left Ventricle ➔ Pumps Blood

- **Systemic Circulation**
- **Through ➔**
- **Aorta (Oxygenated blood)**

Heart Valves

1. **Atrioventricular (AV) valves**

2. **Semilunar valves**

Atrioventricular (AV) Valves

- Present between
- The Atrium and Ventricle
- On both the sides (Right & Left)
- Together called Atrioventricular (AV) Valves

Atrioventricular (AV) Valves

- **Permits flow of blood**
- **From the Atrium To ➔ Ventricle**

Atrioventricular (AV) Valves

- **Prevent ➔**
- **The blood from flowing back**
- **In to the atrium from ventricles**

Atrioventricular (AV) Valves

1. **Tricuspid valve**

2. **Biscuspid valve ➔ Mitral Valve**

Tricuspid valve (RA➔RV)

- 3 Fibrous flaps or Cusps
- Opening between
- Right atrium and Right ventricle
- Guarded by it

Bicuspid valve (LA➔LV)

- 2 Fibrous flaps or Cusps
- Opening between
- Left atrium and Left ventricle
- Guarded by it

Mitral valve (LA➔LV)

- **Mitral valve**
- **Resembles**
- **The Bishop’s headgear termed ➔ Mitrae**

Atrioventricular (AV) Valves

- Opening and closing of the AV valves ➔Passive
- Depends ➔
- Pressure changes in Atrium and Ventricles

Pressure ➔ Increases ➔ Atrium

- AV (Tricuspid & Mitral) valves pushed ➔ Open
- Blood flows from
- Atrium ➔ Ventricles

Contraction of Ventricles

- **Increases the ➔ AV pressure**
- **Closes the AV (Tricuspid & Mitral) valves**

Closure of AV valves

- **Prevents ➔ Backward flow of Blood**
- **From Ventricles ➔ To Atrium**

AV Valves ➔ Held by Papillary Muscles

- Held in position by the ➔Papillary muscles
- Papillary muscles ➔
- Arising from ➔ The walls of the ventricles

Chordae Tendinae

- Attached to ➔ Free margins of AV valves
- With help of ➔ Fibrous strands
- Fibrous strands ➔ Chordae Tendinae

Papillary Muscles → Function

- **Prevent**
- **Bulging of AV valves into the Atrium**
- **Do not help → Opening & Closing of the Valves**

Semilunar Valves

1. **Pulmonary Valve**
2. **Aortic Valve**

Semilunar Valves

1. **Aortic Valve**
2. **Pulmonary Valve**

Semilunar Valves ➔ Termed

- **Semilunar ➔ Semi + Lunar**
- **Cusps have Half-moon shape**

Aortic Valve (LV ➔ Aorta)

- Present between Aorta & Left Ventricle
- Regulate
- Blood flow from Left ventricles ➔Aorta

Pulmonary Valve (RV ➔ PA)

- Present between
- Pulmonary Artery ➔ RightVentricle
- Regulate
- Blood flow from Right ventricles ➔ Pulmonary Artery

Heart Valves ➔When open

- Offer little resistance to flow of Blood
- Small difference in pressure ➔
- Large flow of Blood

In Disease ➔ Heart Valves

- **Narrowed & Not fully open**
- **Increased resistance to the Flow of blood**

In Disease ➔ Heart Valves

- Cardiac muscle
- Has to generate Higher pressure
- To pump the flow of blood

Arteries Carry ➔ Blood

- **Away from the heart**

Veins Carry ➔ Blood

- **Toward the heart**

Arteries Carry ➔ Blood

- All arteries carry ➔ Oxygenated blood
- Except ➔
- Pulmonary Artery (Deoxygenated blood)

Veins Carry ➔ Blood

- Deoxygenated blood ➔ Toward the heart
- Except ➔
- Pulmonary Vein (Oxygenated Blood)

Clinical Physiology

- **Valvular Heart Disease :**
- **A defect or damage to one of 4 heart valves**
- **Mitral (Biscuspid) & Aortic (Semilunar)**
- **Frequently affected**

Valvular Heart Diseases

- **Types ➔**
- **Stenosis**
- **Incompetence**

Valvular Heart Diseases

- **Causes ➔**

1. **Congenital**
2. **Rheumatic Heart Diseases (RHD)**
3. **Bacterial endocarditis (SABE)**

Stenosis → Valvular Heart Diseases

- Valves → Hard
- Passage become →Narrow
- Restricts → Flow of blood out of the contracting chamber

Incompetence → Valvular Heart Diseases

- Valves → Unable to close completely
- Leakage of blood →
- Back into the chamber after it has flown out

Structure of the Wall of the Heart

- **Wall of the heart has 3 layers:**

1. **Epicardium**
2. **Myocardium**
3. **Endocardium**

Epicardium

- Serous layer
- Covering the heart

Myocardium

- **Made up of cardiac Muscle cells**
- ***"Involuntary"***

Endocardium

- Single layer of endothelial cells
- Lining
- Inner surface of the heart

Cardiac Muscles

- **Branching fiber**
- **With**
- **Centrally located nucleus**

Cardiac Muscles

- Adjacent fibers
- Connected by
- Intercalated Discs

Intercalated Disc

- Cell membranes
- That separate Cardiac muscle cells
- From One another

Intercalated Disc

- Electrical resistance through
- Intercalated Disc
- 1/400th of Cardiac cell membrane

Intercalated Disc → Gap junctions

- Specialized intercellular Connection
- Between adjacent fibers
- Offer → Low resistance to the passage of Ions

Functional Syncytium

- Gap Junctions ➔ Because of low resistance bridges
- The cardiac muscle fibers contract
- As Single functional Unit

Desmosomes → Intercalated Disc

- Provide → A site for adhesion
- Between 2 Cardiac muscle cells
- Help to transmit
- The Tension developed in one cell to other during contraction

Functional Syncytium

- 2 Atria
- 2 Ventricles
- Contract simultaneously
- As a single Unit

Cardiac muscle ➔ Contractile Proteins

- Actin and Myosin
- Arranged in a Sarcomere
- It has A-band, I-band
- H-band with M & Z -lines

Cardiac muscle → Sarcoplasmic Reticulum

- **NOT well formed**
- **T- tubule →Wider**
- **T-tubule → Located at the Z-line**
- **T-tubule → Located at A-I junction in the skeletal Muscle**

Force of Cardiac muscle Contraction

- **Depends on ➔**

- **The Muscle load acting on the muscle fiber**

Preload

- If the load acting on the muscle fiber
- Before commencement of the contraction
- Elastic component present in series with contractile element → Stretched

Tension in ➔ Preload

- **Tension in the muscle ➔**
- **Increases till it is able to lift load**
- **Initial part of this contraction ➔ Isometric**

Afterload

- Tension at which
- The load is lifted by muscle
- During Afterload muscle contracts ➔Isotonically
- Without increase in the tension

Nerve supply to the Heart

1. **Sympathetic nerves**

2. **Parasympathetic nerves**

Sympathetic Innervation

- First five thoracic segments of the spinal cord
- T_1 to T_5
- Arise ➔ Stellate Ganglion

Preganglionic Fibers

- Sympathetic Preganglionic fibers
- From T_1 to T_5 Thoracic segments
- Reach Superior, Middle, Inferior cervical ganglion ➔ Sympathetic chain

Postganglionic fibers

- **Arising :**
- **Superior, Middle, Inferior cervical Ganglion**
- **Innervate: Heart**

Noradrenergic Fibers

- **Terminate ➔ Epicardial region**

Sensation from Heart

- **Carried by ➔ Sympathetic fibers**

Cardiac sympathetic Nerve supplies

1. Sinoatrial (SA) node
2. Atrioventricular (AV) node
3. Muscles of Atria
4. Muscles of Ventricles

Stimulation of sympathetic Nerves to Heart

- *Increase* the Heart Rate (HR) ➔
  - Positive Chronotropic effect
- Increase the Force of contraction ➔
  - Positive Inotropic effect

Stimulation of sympathetic Nerves to Heart

- ***Increase*** **the rate of conduction ➔**
  - **Positive Dromotropic effect**
- ***Increase*** **Excitability➔**
  - **Positive Bathmotropic effect**

Mechanism of Action

- **Sympathetic stimulation ➔**
- **Release Epinephrine (Adrenaline)**

Epinephrine ➔ Inotropic Effect

- Epinephrine ➔ β_1-adrenergic receptors
- Activates ➔Adenyl cyclase
- Enhances ➔ Intracellular cAMP
- cAMP ➔ Opening of Long lasting Ca^{2+} Channels
- Influx of ➔Ca^{2+} ions

Epinephrine ➔ Chronotropic Effect

- Increasing Influx of Ca^{2+} ions ➔ In to SA node
- Increase ➔Rate of spontaneous depolarization
- Reduce ➔ Efflux of K^{+} ions

Epinephrine ➔ AV node

- **Increases rate of impulse transmission**
- **Through AV node**

Clinical Physiology

1. Glucagon
2. Caffeine & Theophylline
3. Digitalis
4. Quinidine, Procainamide, Barbiturates
5. Hypoxia, Hypercapnia, Acidosis

Glucagon ➔ Positive Inotropic effect

- **Increases formation of cAMP**

Caffeine & Theophylline

- **Positive *Inotropic* effect**
- **Prevent breakdown of cAMP**

Digitalis ➔ Positive Inotropic effect

- **Inhibiting Na^+ - K^+ ATPase pump**
- **In myocardium**

Myocardial Contractility

- **Reduced by :**

1. **Quinidine**
2. **Procainamide**
3. **Barbiturates**
4. **Hypoxia, Hypercapnia, Acidosis**

Parasympathetic Innervation

- Through ➔ Vagus nerve (X^{th} CN)
- Preganglionic Parasympathetic fibers ➔
- Vagus nucleus

Parasympathetic Innervation

- Preganglionic Parasympathetic fibers ➔
- Vagus nucleus
- Pass through ➔ Cardiac nerve
- End in ➔ Parasympathetic ganglion in the heart

Parasympathetic Innervation

- Postganglionic Parasympathetic fibers ➔
- Parasympathetic ganglion in the heart
- Innervate ➔ Heart

Noradrenergic Fibers

- **Terminate ➔ Epicardial region**

Vagal fibers

- **End in ➔ Endocardial region**

Right Vagus fibers ➔ Supply

- **SA (Sinoatrial) node ➔**
- **Pacemaker**

Left Vagus fibers ➔ Supply

- **AV (Atrioventricular) node**

No ➔ Parasymapathetic supply

- **Ventricles**

Stimulation of parasympathetic Nerves to Heart

- ***Decrease*** **the Heart Rate (HR) ➔**
  - **Negative Chronotropic effect**
- ***Decrease*** **the Force of contraction ➔**
  - **Negative Inotropic effect**

Stimulation of parasympathetic Nerves to Heart

- ***Decrease*** **the rate of conduction ➔**
  - **Negative Dromotropic effect**
- ***Decrease*** **Excitability➔**
  - **Negative Bathmotropic effect**

Athletes➔Increase in Vagal Tone

- **Increase in Vagal Tone**
- **Reduces ➔ Resting heart rate (HR)**

Mechanism of Action

- **Stimulation of Vagus ➔ Release Acetylcholine**
- **Acetylcholine acts on ➔**
- **Muscarinic receptors**

Ach ➔ Decrease Heart Rate

- Acts ➔ Through ➔ G-Proteins
- Opens ➔ K^+ ion channels
- Promotes ➔Efflux of K^+ ions
- Cause ➔ Hyperpolarization of membrane
- Decreases ➔ Slope of Action Potential
- Reduces ➔Rate of impulse generation in SA node

Left Vagus fibers ➔ AV node

- Stimulation ➔ Left vagus
- Delays conduction of the impulse through
- AV node
- Negative dromotropic

Ach → Reduce force of cardiac muscle contraction (Negative inotropic)

- Reducing → Intracellular cAMP
- Delaying → Opening of Ca^{2+} ion channels
- Reducing → Influx of Ca^{2+} ions
- Reduce force of contraction
- Negative Inotropic

Strong stimulation of Vagus

- **Stops ➔ Heart for short duration**

Vagal escape

- **Heart overcomes ➔**
- **Inhibitory effect on prolonged vagal stimulation**

Vagal escape ➔ Cause

- The Atrioventricular node (AV node)
- Taking over generation of impulse
- After the inhibition of the SA node (Pacemaker)

Moderate tonic discharge

- **Cardiac sympathetic nerves**
- **At rest**

Vagal Tone ➔

- **Considerable**
- **Tonic vagal discharge (Vagal tone)**
- **In humans and other large animals**

Explain what will happen & why…?

- After the administration of
- Muscarinic receptor antagonists
- Such as Atropine

Explain what will happen & why…?

- The heart rate in humans
- Increases from 70 beats/min
- Its normal resting value,
- To 150–180 beats/min

Explain what will happen & why…?

- **Because**

- **The sympathetic tone is unopposed**

Noradrenergic & Cholinergic blocked

- **The heart rate is approximately 100 beats/min**

Clinical Physiology

- **Connections ➔**
- **Reciprocally inhibit ➔**
- **Sympathetic and Parasympathetic systems**

Clinical Physiology

- **Acetylcholine (Parasympathetic systems)**
- **Acting in Presynaptic regions**
- **Reduces release of**
- **Norepinephrine (Sympathetic system)**

Clinical Physiology

- **Neuropeptide Y ➔**
- **Inhibits release of Acetylcholine (Parasympathetic systems)**
- **From noradrenergic endings**

Properties of Cardiac Muscles

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Properties of Cardiac Muscles

1. Autorhythmicity
2. Excitability
3. Conductivity
4. Refractory period
5. All-or-none law
6. Tone
7. Contractility
8. Staircase phenomenon
9. Extrasystole with compensatory pause

1. Autorhythmicity

- Ability of the cardiac muscles
- To generate their own impulses
- Called ➔ Autorythmicity
- Without external stimulation

2. Excitability

- **Responding property**
- **of the tissue**
- **To a stimulus**

Action potential in Ventricular Muscle

- **Averages ➔**
- **About -105 millivolts**

RMP in Ventricular Muscle fiber

- – 80 to – 85 milliVolts

RMP ➔ Myocardial Fibers

- **Of approximately ➔ −90 mV**
- **[Ganong Review of Medical physiology 25th Edition Chapter 29 Pg no:521]**

Intracellular Potential Rises ➔ + 20mV

- From a very negative value ➔
- About −85 millivolts
- Between Heart beats
- To a slightly positive value ➔ During Each beat
- About +20 millivolts

Plateau

- **The excited membrane**
- **“Does not ➔Repolarize”**
- **Immediately ➔ After depolarization**

Potential remain on a Plateau

- Near the peak of the spike potential
- For many milliseconds

Plateau last for ➔ 0.2 - 0.3 sec

- And causes
- Contraction of heart muscle
- To last
- For this same long period

After Initial *Spike* ➔ Membrane

- **Remains ➔ Depolarized**
- **For about ➔0.2 second**
- **Exhibiting a “*plateau*”**

Cause of Plateau

- **Combination of several factors**
- **In heart muscle**

Heart ➔Two types of channels contribute to the depolarization process

1. *Voltage-activated sodium (Na^+)channels*
2. *Voltage-activated calcium-sodium (Ca^{2+} - Na^+) channels*

Voltage activated Na^+ Channel

- *Usual channel*
- *"Fast channel"*

Voltage activated Na^+ Channel

- ***Opening* of ➔ *"fast channels"***
- **Causes**
- **The *spike portion of the action potential***

Voltage activated Ca^{2+}- Na^{+} Channel

- *"L-type calcium channels"*
- **Which are**
- **Slow to open called ➔ "*Slow channels*"**

Voltage activated Ca^{2+}- Na^{+} Channel

- ***Prolonged opening (Slow Channel)***
- **Mainly allows**
- **Calcium (Ca^{2+}) ions to enter ➔ The fiber**

Voltage activated Ca^{2+}- Na^{+} Channel

- **Largely responsible ➔**
- ***The plateau portion of the action potential***

Role of Voltage gated K^+ Channel

- *Slower opening of Voltage gated K^+ Channel*
- **Delays ➔ The return of the membrane potential**
- **Toward its Normal negative value**
- **Of −80 to −90 millivolts (RMP)**

End of Plateau when➔

- **“Calcium-Sodium channels”**
- **Slow Channels ➔ Close**
- **And**
- **Permeability to Potassium ions ➔ *Increases***

Importance of *Plateau*

- Greatly prolongs the period of depolarization
- Ventricular contraction to last as much as
- 15 times as long in ➔ Cardiac muscle
- As in skeletal muscle

Plateau F/b ➔ Repolarization

- **At the end of the plateau**
- **Action potential followed**
- **By abrupt *Repolarization***

Phases of Cardiac Muscle Action potential

1. *Phase 0 (depolarization)*
2. *Phase 1 (initial repolarization)*
3. *Phase 2 (plateau)*
4. *Phase 3 (rapid repolarization)*
5. *Phase 4 (resting membrane potential)*

Phase 0 → Depolarization

- *Fast sodium channels → Open*
- **When the cardiac cell is → Stimulated**
- **And depolarizes**
- **Membrane potential becomes → More positive**

Phase 0 → Depolarization

- Voltage-gated sodium channels
- Fast sodium channels→ Open
- Permit Sodium → To rapidly flow into the cell
- And depolarize

Phase 0 → Depolarization

- **The membrane potential**
- **Reaches about +20 millivolts**
- ***Before the sodium channels close***

Phase 1 → Initial Repolarization

- *Fast sodium channels → Close*
- **The cell begins → To repolarize**
- **And K^+ ions →Leave the cell**
- **Through → Open potassium (K^+) channels**

Phase 2 ➔ Plateau

- **A brief initial repolarization**
- **There occurs plateaus**

Phase 2 ➔ Plateau

- *Calcium (Ca^{2+}) channels ➔ Open*
- *Fast potassium (K^{+}) channels ➔ Close*

Phase 2 ➔ Plateau

- **Increased ➔ Calcium ion permeability**
- ***Increased calcium ion influx***
- **Decreased ➔ Potassium ion permeability**
- ***Decreased potassium ion efflux***

Phase 3 ➔ Rapid Repolarization

- *Calcium (Ca^{2+})channels ➔ Close*
- *Slow potassium (K^{+}) channels ➔ Open*

Phase 3 ➔ Rapid Repolarization

- **The closure of calcium *(Ca^{2+})* ion channels**
- ***Increased potassium (K^{+}) ion permeability***
- **Permitting**
- ***Potassium (K^{+}) ions to rapidly exit the cell***

Phase 4➔Resting Membrane Potential

- Ends the plateau
- Returns the cell membrane potential
- To its resting level (-80 to -90 mV)

Phase 4➔Resting Membrane Potential

- *Averages about ➔ −90 millivolts*

Phase 4➔Resting Membrane Potential

- ***Ionic balance is restored by***
- ***Sodium – Potassium ATPase pump***
- ***Sodium – Calcium ATPase pump***

Duration of Cardiac muscle Action Potential

- **200 – 250 ms**

Action potential in Atrial Muscle

- **Ionic basis ➔ Same as in Ventricular Muscle**
- **Duration of Plateau phase (Phase 2) ➔ Shorter**
- **Repolarization phase (Phase 3) is ➔ Prolonged**

3. Conductivity

- Impulses generated by
- Pacemaker tissue → SA node → Sinoatrial node
- Spread to the entire Cardiac muscle with
- Conducting system of the heart
- Junctional tissue

3. Conductivity

- Presence of Gap-Junctions (Desmosomes)
- Between the cardiac muscle fibers
- Hasten ➔ Movement of Ions
- Rapid spread of impulse

4. Refractory period

- Cardiac muscle
- Like all excitable tissue is
- Refractory to re-stimulation during the action potential

Normal Refractory period of Ventricles

- 0.25 to 0.30 second
- Which is about the
- Duration of the prolonged plateau action potential

Absolute Refractory Period

- **0.25 to 0.30 second ➔ Period during**
- **Which the 2nd stimulus of**
- **Any intensity ➔ Fails to produce a response**

Absolute Refractory Period

- Correspond to Major part ➔
- Long plateau phase ➔
- Of Cardiac Action potential

Cardiac muscle Can’t be Tetanized

- **Because of**
- **Long Absolute Refractory period**
- **0.25 to 0.30 seconds**

Relative Refractory Period

- **Additional**
- ***Relative refractory period of about***
- ***0.05 second***

Relative Refractory Period (0.05 sec)

- During which the muscle is ➔ *More difficult*
- To excite than normal but nevertheless
- *Can be excited* by a *very strong excitatory signal*

Relative Refractory Period (0.05 sec)

- 2nd stimulus of Greater intensity
- Produce a ➔ Response ➔
- Demonstrated by ➔ Premature contraction

Relative Refractory Period

- **Corresponds to**
- **Last part of the repolarization**

Refractory Period of ➔ Atria

- Much Shorter than Ventricular muscle
- About 0.15 second ➔ For the Atria
- 0.25 to 0.30 second ➔For the Ventricles

5. All-or-None Law

- **When the cardiac muscle ➔ Stimulated**
- **With “Threshold” stimulus**
- **It responds to its Maximum**

5. All-or-None Law

- **When the cardiac muscle ➔ Stimulated**
- **Is Strength of Stimulus is ➔ “Subthreshold”**
- **The muscle ➔ Does not show any response**

5. All-or-None Law

- **Applies to**
- **The entire Cardiac muscle**

6. Tone

- **Partial contracted state of myocardium**
- **Without**
- **An external stimulus**

7. Contractility

- **Chemical changes**
- **Mechanical shortening of the muscle**
- **In response to a stimulus**

7. Contractility

- **Molecular basis**
- **For the cardiac muscle contraction**
- **Is similar to that of the skeletal muscle contraction**

8. Staircase Phenomenon

- **Heart is stopped by ➔**
- **Stimulation of the ➔ Vagus nerve**
- **X^{th} - CN ➔Parasympathetic**

8. Staircase Phenomenon

- Heart is stopped by ➔ Vagal Stimulation
- Later,
- Heart is allowed to ➔ Beat again
- By withdrawing ➔ Vagal stimulation

Withdrawing ➔ Vagal Stimulation

- **Initial few contractions**
- **Shows**
- **An increase in the amplitude of contractions**
- **Termed as ➔ *Staircase phenomenon***

1st Cause of Staircase Phenomenon

- Accumulation of Ca^{2+} ions released
- During the previous contraction
- Potentiating
- The effect of subsequent contractions

2nd Cause of Staircase Phenomenon

- Decrease in the Viscosity of the muscle

3rd Cause of Staircase Phenomenon

- Increase in temperature of the muscle

3rd Cause of Staircase Phenomenon

- Increase in temperature of the muscle
- Enhances the enzymatic activity
- Improves contraction

9. Extrasystole with Compensatory pause

- When 2nd the stimulus
- Falls during any part of systole
- It has no effect
- Heart continuous to beat as before

9. Extrasystole with Compensatory pause

- When 2nd the stimulus
- Falls during Diastole
- The heart ➔ Contracts immediately

9. Extrasystole with Compensatory pause

- This extra contraction called “Extrasystole”
- or Premature beat is followed by
- A pause called the
- “Compensatory pause”

Starling's Law

- The force of contraction
- Is directly proportional
- To the initial length of the muscle fiber
- Within physiological limit

Starling's Law in respect to Heart

- Greater the Preload (Venous Return)
- Greater the force of contraction
- Within physiological limit

Importance of Starling's Law

- Helps to maintain the Cardiac Output
- Equally
- To both Right and Left sides of the heart

Importance of Starling's Law

- In Case of Heart Failure
- The heart pumps out excess blood
- Which has accumulated during earlier contractions
- To reduce load on the heart

Molecular basis of Starling's Law

- When muscle ➔ Stretched
- Actin & Myosin are ➔ Separated
- Exposing ➔ Active binding sites
- On the Actin molecules

Molecular basis of Starling's Law

- Exposing Active binding sites
- Increases the number of cross-bridges formed

Molecular basis of Starling's Law

- Greater the number of cross-bridges formed
- Greater the force of contraction

Molecular basis of Starling's Law

- Actin filament moves
- A longer distance in stretched muscle
- During contraction ➔
- Increase the force of contraction

Molecular basis of Starling's Law

- When muscle stretched ➔Preload
- Resting metabolism ➔Increases
- Increase in temperature of the muscle
- Better enzymatic activity ➔
- Better force of contraction